Methods for Reduction of Sternal Wound Infection

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Deep sternal wound infections continue to be an uncommon but potentially devastating complication of cardiac surgical procedures. Numerous risk factors have been identified but only a few can be characterized as modifiable. These risk factors and their modifications are reviewed in the following article.

Although the overall incidence of postoperative deep sternal wound infections after heart surgery continues to be low, the prevention and subsequent management of the complication still poses a daunting task for every cardiac surgeon. The reported incidence ranges from 1.3% to 5% in most recent series and with a high associated mortality rate of 9.8% to 14%.[1-3] In addition to the increased mortality, the condition also carries with it a high morbidity, resulting in prolonged hospitalization and the associated increase in cost. The average cost of hospitalization for a patient with a sternal infection is approximately three times that of patients without wound difficulties.[4]

Postoperative sternal infection exists along a spectrum of presentations from sterile wound dehiscence to suppurative mediastinitis, and its pathogenesis is complex and multifactorial. However, a number of specific patient and procedure related risk factors have been delineated.[5-8] These include obesity, diabetes, reoperation, increased duration of the operation, coronary artery bypass grafting, use of bilateral internal thoracic arteries, postoperative inotropic support, and the need for multiple blood transfusions. While most of these risk factors are not amenable to intervention, others can be modified with a consequent reduction of sternal wound complications. The purpose of this review is to attempt to identify and highlight a number of these modifiable risk factors: antibiotic prophylaxis; preoperative preparation; intraoperative management; and sternal closure techniques.

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Microbiology

The microbial etiology of sternal wound infections can be varied and include Gram-positive and Gram-negative bacteria as well as fungi. However, numerous recent studies have demonstrated that the most common causative pathogens involved in sternal wound infections are *Staphylococcus epidermis* and *Staphylococcus aureus*, both from the normal flora of the skin.[9-11] That these organisms account for over half of postoperative mediastinitis suggests that skin flora, introduced into the wound at the time of operation, is a major and potentially modifiable risk factor.[9,12]

Often regarded as a relatively benign organism, *S. epidermis* (coagulase-negative staphylococcus) has emerged as the most common sternal infection pathogen and often presents with only minimal signs of systemic infection. It often has a slow and late onset of up to 3 weeks.[12] Interestingly, this organism appears more frequently in association with sternal instability.[7,9] The proposed mechanism for this association is that the deep sternal infection originates from a minor cutaneous or subcutaneous infection and then spreads into the mediastinal space when sternal dehiscence disrupts the mechanical barrier between the mediastinum and the presternal tissues. A superficial, self-contained pocket of infection with coagulase-negative staphylococci that would otherwise be benign can thus progress into a deep infection.

S. aureus is the other major pathogen. It has been increasingly associated with colonization of the nasal passages of patients, leading to specific dissemination in the operating room.[10,13]

Another major group of bacteria involved in the genesis of deep sternal infections is aerobic Gram-negative rods. The etiology of these infections is likely to be entirely different from those caused by staphylococci. These Gram-negative mediastinal infections are associated with a more complicated postoperative course and concomitant nosocomial infection.[6,9] These include pneumonia from prolonged mechanical ventilation, urinary tract infections, and intraabdominal sepsis. Aggressive treatment of these comorbid conditions should also reduce the risk of mediastinitis.

Antibiotic Prophylaxis

The perioperative administration of antibiotics is a universal but not extensively studied component of prophylaxis against sternal wound infections. Two significant conclusions form the basis of most prophylactic antibiotic regimens.[14] First, antibiotic prophylaxis significantly reduces the prevalence of sternal infections when compared with placebo. Second, increasing the duration of prophylaxis beyond 36 to 48 hours does not further reduce sternal infection rates. The 1999 consensus paper from the Hospital Infection Control Practices Advisory Committee (HICPAC) recommends treatment consistent with those two conclusions.[15] Common practice in many hospitals is to use a second-generation cephalosporin, with vancomycin substituted for it in penicillin-allergic patients or for valve operations, and modulated by hospital-specific antibiotic sensitivities.

The incidence of nasal colonization with *S. aureus* in the normal population is reported to range from 10% to 15%.[11] Such colonization increases the risk of sternal wound infections.[11,13,16] Proposed mechanisms for the transfer of nasal *S. aureus* to the sternal wound include direct spread from the nose to hands to the incision, and hematogenous spread caused by endotracheal tube trauma to the nose.[13] Elegant DNA fingerprint analysis demonstrates the genotype of *S. aureus* isolates recovered from the patient's sternum and nose often were identical. This observation led to the use of topical nasal antibiotics in the preoperative and early postoperative period in attempt to eliminate the *S. aureus* and reduce the risk of sternal infection.[13,17] Perioperative application of nasal mupirocin eradicates 95% to 100% of *S. aureus* up to 1 year postoperatively,[18] and demonstrates a 67% reduction of infection in a mupirocin treated group as compared with a retrospective control group.[13]

Hyperglycemic Control

In diabetic patients, it is has been demonstrated that aggressive and intensive blood sugar control is associated with improved survival following cardiac operations. Diabetes mellitus *and* perioperative hyperglycemia have been identified as independent risk factors for deep sternal wound infections.[19,20] The study by Furnary and coworkers demonstrated that continuous insulin infusion (glucose kept in range 150-200 mg/mL) when compared with intermittent subcutaneous insulin, led to a significant reduction in the incidence of deep sternal wound infections (0.8% versus 2%).[21]

Sternal Stability and Closure Techniques

Numerous studies now support the emerging concept that sternal instability is a major risk factor in the development of sternal wound infections.[9,10,22,23] It is postulated that this increased infection rate seen with unstable sternal fixation is due to increased bone movement, which damages local tissue, leads to tissue necrosis, and creates a milieu for bacterial growth at the time of transient inoculation.[9,24] In contrast, stable bony fixation reduces the amount of tissue trauma and promotes more rapid vascularization and primary bone healing.

A careful and precise midline sternotomy reduces the risk of later sternal instability and dehiscence.[23] A paramedian sternotomy predisposes to sternal instability regardless of the closure technique.

The orthopedic surgery literature documents that fracture instability increases the risk of infection. In a study by Worlock, rabbit tibial fractures were stabilized with either a dynamic compression plate (stable group) or a loose intramedullary rod (unstable group).[24] These fractures were then inoculated with *S. aureus*. The

infection rate was double in the unstable group (71% versus. 35%).

In primates, Sargent and colleagues, tested the hypothesis that rigid fixation enhanced sternal bone healing.[25] They compared interrupted wire suture technique with compression miniplates (their model for rigid fixation) in skeletally mature baboons. By carefully studying sternal harvests from each group, they were able to show that at 4 weeks, clinical stability was superior with rigid fixation and resulted in increased new bone formation across the osteotomy gap. Rigid fracture stabilization promulgates earlier bone healing and therefore greater resistance to infection.

Multiple techniques of sternal closure include the use of simple cerclage wires, figure-of-eight wires, figure-of-eight stainless steel cables, stainless-steel plates, sternal screws with a central lumen for wire placement, bone staples to buttress cerclage wires, thermoreactive clips, and intercostal weaving of wires to buttress cerclage wires.[22,26-32] Most studies demonstrate the simple peristernal cerclage technique to be the least stable (but most widely used) method of sternal approximation. Since the goal of every closure method should be to achieve sternal rigidity and strength, we believe that greater emphasis should be placed on this critical portion of the operation, with adoption of alternative closure methods to cerclage wires.[22]

Preoperative and Intraoperative Techniques

A number of simple techniques should be employed perioperatively to reduce the risk of sternal infection. The routine use of skin shaving using electrical clippers has been shown to be superior to shaving using a razor blade in reducing mediastinitis.[33] Blade shaving is believed to lead to bacterial contamination by creating microscopic lacerations in the skin.

Another risk factor implicated in sternal infections is the indiscriminate use of electrocautery for hemostasis in the presternal soft tissues.[34] This compromises tissue viability and leaves protein char, thus predisposing to bacterial inoculation. Therefore, meticulous pinpoint electrocautery to achieve hemostasis when dividing the soft tissues is recommended.

The routine application of bone wax as a hemostatic agent following sternotomy has also been implicated in promoting sternal infections with a reduced bacterial inoculum required to produce sternal osteomyelitis in the presence of bone wax.[35] This infection predilection appears to be caused by local inhibition of osteogenesis and intense local inflammation. An alternative to bone wax is the use of topical vancomycin and powdered absorbable gelatin (Gelfoam®) mixed with topical thrombin to form a hemostatic paste. When applied to cut sternal edges, this paste was not only hemostatic but was also shown to reduce postoperative sternal infection rates.[36]

Summary

Multiple practices and techniques easily can be altered to reduce the incidence of sternal and mediastinal infections.

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